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Cadmium in blood and urine after cessation of exposure

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ABSTRACT

A plant for the synthesis and packaging of cadmium oxide was operated for 32 months (1982–84) in a small chemical factory producing zinc and copper oxide. The cumulative exposure of 6 workers was from 12 to 190 days. Five years after cessation of exposure the blood cadmium levels in the exposed were, on average, 4–10 times higher than those of a reference group and the urinary cadmium levels were 1–6-times higher. In view of the long half-life of cadmium in the human body, the choice of normal reference values requires particular care since brief and sometimes forgotten exposures to cadmium may be a confounding factor to set reference values.

Key words: cadmium; blood; urine; cessation of exposure

INTRODUCTION

A small chemicals factory producing zinc and copper oxide operated a plant for the synthesis and packaging of cadmium oxide for 32 months, between 1982 and 1984.

Production was on a monthly basis, 1 week each month, the cycle lasting about 12 h. A 1000–1200-kg quantity of cadmium blocks were melted in a crucible and the metal vapours, transformed into cadmium oxide on contact with air, were conveyed to a hopper for manual packaging in drums.

The working environment had no exhaust ventilation system and the area was cleaned manually using brooms. The workers were allowed to smoke and eat at the work place and protective clothing was kept with ordinary clothing.

During the first 2 years of operation of the 'cadmium department' the workers never underwent any medical examinations for the specific risk and were only sent to our unit for the first time in October 1984. The results of the absorbed dose indicator tests were so high that the factory discontinued cadmium oxide production before any industrial hygiene survey could be carried out.

MATERIALS AND METHODS

Subjects

The population under study consisted of 2 workers who participated in all the monthly production campaigns so that they had a cumulative exposure of 190 days: a third worker participated for about 90 days, 3 others were exposed for not more than 12–18 days each, and a reference group composed of 8 employees of the same factory not exposed to cadmium. Biological monitoring was continued after cessation of exposure up to the present for a total of 60 months. For the first control, the 3 workers with highest exposure had such high blood cadmium values that hospitalization in the renal unit was advised; however, blood chemistry tests excluded the presence of a renal impairment.

Biological measurements

Periodic checks for all exposed and non-exposed subjects consisted of blood count, creatinemia, creatinuria, urine analysis, cadmium, zinc and copper in blood and urine, urinary β_2-microglobulin, respiratory function and chest X-ray. Blood samples were obtained by venipuncture using cadmium free disposable syringes and collected in EDTA cadmium free tubes.

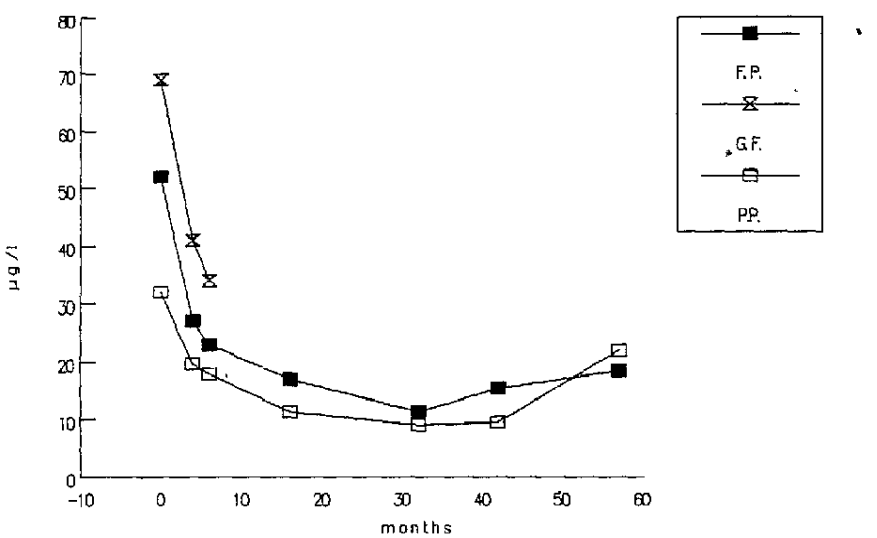

Fig. 1. Cd-B follow-up of three workers after exposure.

An early morning spot urine sample was collected in a bottle and adjusted to pH 7 to avoid degradation of β_2-microglobulin.

The analyses of Cd in whole blood (Cd-B) or urine (Cd-U) were performed by electrothermal atomic absorption spectrometry (Perkin Elmer Zeeman 3030-HGA600) and with an AS60 autosampler. Internal quality control was carried out with the Standard Community Bureau of Reference (E.E.C.) BCR 194–195 for lower and higher cadmium concentrations.

RESULTS

The evolution of urinary and blood cadmium in the 2 workers with a total exposure of 190 days (F.P. and G.F.) and the third worker with an exposure of 90 days (P.P.) is shown in Figs 1 and 2.

Controls made 5 and 7 months after cessation of exposure revealed a rapid drop in Cd-B values, by about half, from 69, 52 and 32 μg/l to 41, 27 and 20 μg/l. Cd-B subsequently decreased slowly until the 30th month (11 and 9 μg/l) and then rose in the following 30 months (18 and 22 μg/l).

Cd-U values, however, did not change in the first 7 months being constantly around 70, 60 and 20 μg/l; the values then slowly decreased up to the 30th month and then remained unchanged until the 60th month.

β_2-microglobulin showed wide variation with values exceeding 300 μg/l, and were apparently, though roughly, correlated with the Cd-B and Cd-U values.

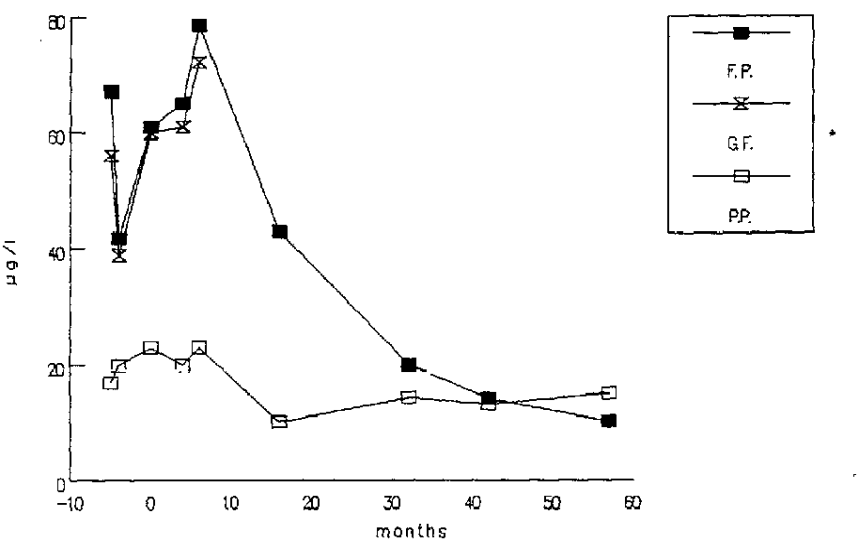

Fig. 2. Cd-U follow-up of the three workers after exposure.

The number of samples analysed for urinary β_2-microglobulin were sufficient to permit a statistical analysis: β_2-microglobulin concentrations varied with the values of the biological exposure indicators but the variations were not statistically significant either between classes or compared with the reference group. In the reference group, the Cd-B, Cd-U and β_2-microglobulin mean values were 1.70 μg/l, 2.2 μg/l and 178 μg/l respectively.

Five years after cessation of exposure, Cd-B in the 3 classes, (according to exposure of 190, 90 and 18 days), was 17, 15.8 and 7.3 μg/l, respectively. Cd-U levels were 12.1, 14.1 and 4 μg/l, and β_2-microglobulin levels were 245, 217 and 210 μg/l, respectively. From this Cd-B values in the exposed subjects were calculated to be 4–10-times higher and Cd-U were found to be 1–6 times higher.

DISCUSSION

In the absence of industrial hygiene data, the assumption that the workers had been exposed to an extremely high risk of cadmium intoxication via inhalation and probably via the oral route too, was proven by the very bad hygiene conditions observed on inspection of the factory and by the elevated values of the indicators of absorbed dose, which are among the highest reported in the literature [1].

In view of the absence of biochemical and clinical signs of intoxication, two hypotheses can be suggested. The first is that exposure to the metal, although intense, was not sufficiently prolonged over time so as to reach critical concentrations in the target organ, i.e. the renal cortex [2–5]. The second is based on the fact that the 3 workers were simultaneously exposed to zinc oxide, as shown by the high values of zinc in blood and urine, one of them even had a sub-acute zinc intoxication in 1981 [6]. It is therefore likely that the excess of zinc interfered with the toxic action of cadmium via a competitive mechanism towards the enzymatic systems which control reabsorption and protein catabolism in the kidneys [7].

The data reported cover a very small number of subjects and need to be confirmed by studies in larger populations. We recommend that, in view of the long half-life of cadmium in the human body, particular care be taken in the choice of reference values, since brief and sometimes forgotten exposures to cadmium can still influence the results after several years; so 'cessation of work' may be a confounding factor in the setting of reference values for Cd-B and Cd-U.

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